



Are Health Effects Associated with Particulate Matter Near Southern California Freeways?

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research
and
development

Science Question/Background

Mobile sources emissions and their associated health effects represent a **priority area for research**, particularly in the Los Angeles Basin. Past studies have demonstrated health impacts for persons living in **close proximity to roadways** and further evidence suggests that people working and going to school near roads with heavy traffic may also have an increased risk of **adverse health effects** associated with exposure to mobile source pollution. Driving in **bumper to bumper traffic** leads to in-vehicle concentrations of pollutants that are up to **10 times** higher than those in ambient city air; in-vehicle average particle number concentrations on busy freeways can be as high as 543,000 particles/cc.

Research has demonstrated a **linkage** between **exposure to motor vehicle emissions** and **respiratory health**. Additionally, the pollutants associated with these emissions are suspected of **affecting neurologic, immune and developmental systems**. Advances in geographic information systems (GIS) and aerosol concentration technologies have allowed investigators additional means of clarifying the relationship between exposure to traffic related emissions and health.

Given the complex nature of the issue, a variety of methodological approaches have been utilized to explore exposure to mobile source emissions and health effects, each focusing on specific health endpoints.

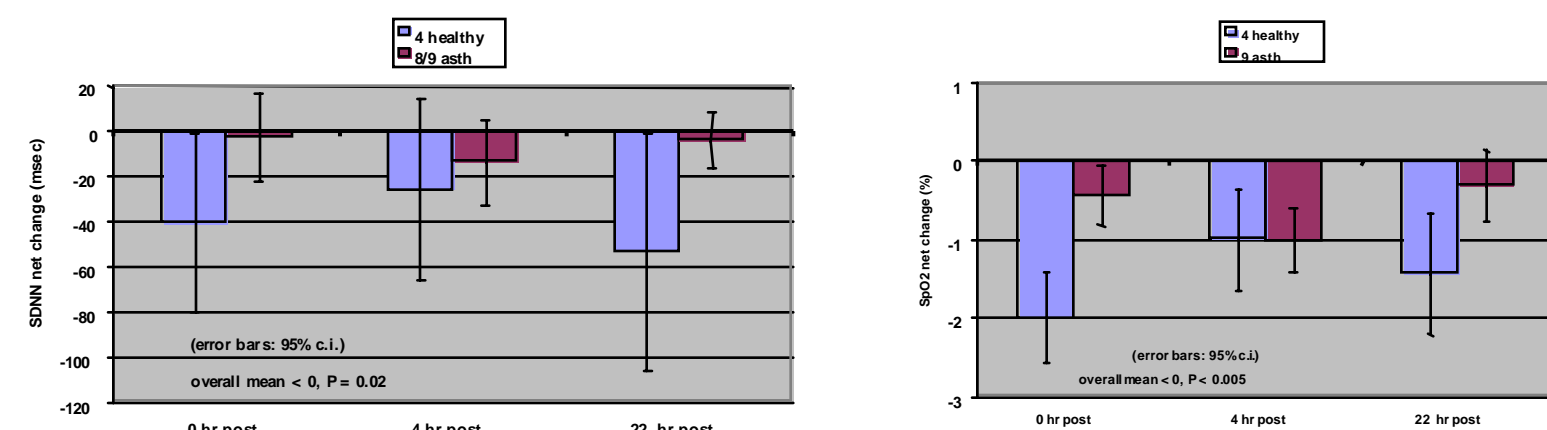
The approaches highlighted here are:

- **Human** clinical studies investigating acute cardiopulmonary responses to short term exposures to concentrated ambient particles
- Long-term **epidemiologic** studies focusing on the effects of chronic air pollution exposures on respiratory health of children
- Utilizing **GIS modeling** and birth records to determine the influence of distance weighted traffic density on pre-term and low birth weight babies
- **Animal toxicology** studies exploring the effects of near freeway exposure on allergic airway disease response, inflammatory effect of inhaled PM on the central nervous system, and cardiovascular effects.

Human Clinical Exposures to CAPs in Los Angeles

This study's hypothesis is that **short-term exposures** to concentrated ambient particles (**CAPs**) in LA will cause **acute cardiopulmonary responses** which may explain epidemiologic findings. Researchers conducted ultrafine and filtered air (FA) **exposures** of volunteers (healthy, asthmatic).

Methods: **2 hour** exposures; Moderate **exercise** for 15 minutes each half hour; Ultrafine and FA exposures occur in random order and at least 2 weeks apart. **Measures of response:** Lung function tests; symptom questionnaires; blood pressure; arterial O₂ saturation; Holter electrocardiogram; assays of blood and induced sputum, cell counts, mediators of inflammation, coagulation.



SDNN measured during 5-min intervals of rest (0/4/22 hr after the end of a 2-hr UF exposure) showed a significant response. The graph shows group mean differences for healthy and asthmatic subgroups at each time of measurement. The overall mean difference was significantly below zero.

The % O₂ in arterial blood as estimated by fingertip pulse oximeter was significantly reduced. The graph shows net change (UF exposure - FA control) for each subgroup at time of measurement.

Conclusions: Changes in **heart rate variability** were observed following exposure to CAPs. Lung function disturbances were noted (data not shown). The reduction of O₂ in arterial blood may have clinical significance in **susceptible individuals** with **compromised cardiopulmonary** status.

Children's Health Study

The Children's Health Study began in 1993 and is a large, longitudinal study of the effects of **chronic air pollution** exposures on the **respiratory health of children** living in Southern California. It has been funded by a number of agencies, including NIH, CARB, and the EPA.

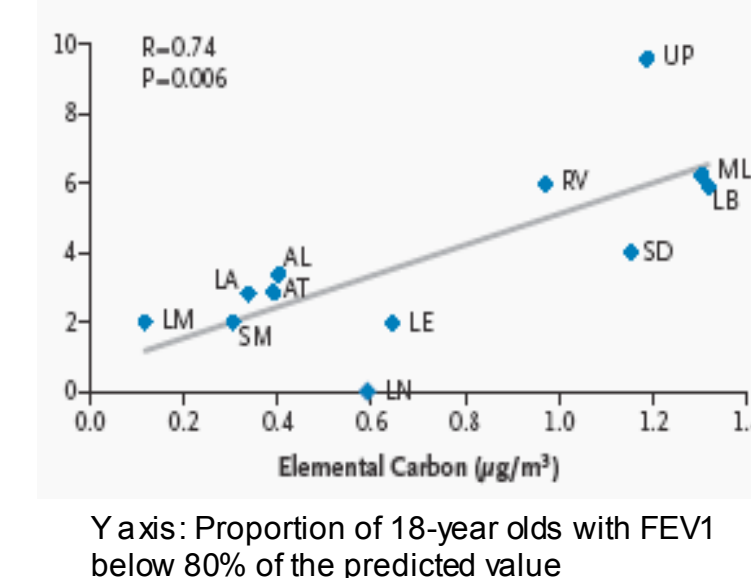
Methods: About **5500 children** in twelve communities have been enrolled in the study. Data on the children's health, their exposures to air pollution, and many **factors that affect their responses** to air pollution were gathered annually starting with the fourth grade and continuing until they graduate from high school. The **twelve communities** in the study were chosen because they have different patterns of high and low levels of the four pollutants: ozone, nitrogen dioxide, acid vapor, and particulate matter.

Association of asthma with exposure to traffic at home

Exposure metric	OR ^a	(95% C.I.)
Measured NO ₂ ^a	1.83	(1.04, 3.21)
Distance to Freeway	1.89	(1.19, 3.02)
Traffic volume within 150 meters	1.45	(0.73, 2.91)
Model-based pollution from:		
Freeways	2.22	(1.38, 3.63)
Non-freeways	1.00	(0.75, 1.33)
Freeways and non-freeways	1.40	(0.88, 2.27)

^a Odds ratio per change of one interquartile range (IQR). For NO₂ this is given as the OR per 5.7 ppb; for distance to freeway, as the OR for the 25th percentile compared to the 75th percentile, (i.e. living closer compared to farther from the freeway). For remaining traffic variables, it is the OR for the 75th percentile compared to the 25th percentile. All models were adjusted for sex, race, hispanic ethnicity, cohort, and community.

Lung development and exposure to air pollution



Conclusions: **Asthma prevalence** in the Children's Health Study is associated with residential distance to freeways, both within and across communities. Current levels of air pollution associated with mobile sources have chronic, adverse effects on **lung development** from the age of 10-18 years leading to **clinically significant deficits** in attained FEV1 as children reach adulthood.

Traffic Density and Pre-term and LBW Babies

Studying **pregnancy** and **exposure to air pollution** is particularly important as developing fetuses are uniquely **sensitive to environmental toxins**. This study estimates **traffic-related birth outcomes** using existing data for large areas in the Los Angeles Basin.

Methods: **Distance weighted traffic density (DWTD):** Traffic count on all streets within a certain radius weighted by distance from road; **Air dispersion model:** Incorporates emission levels, road geometry, meteorology. For 1994-96, in 112 LA zip codes, all LBW and preterm infants (N=31,191) were identified along with a random sample of controls (~ same N); residential birth addresses were then mapped using GIS.

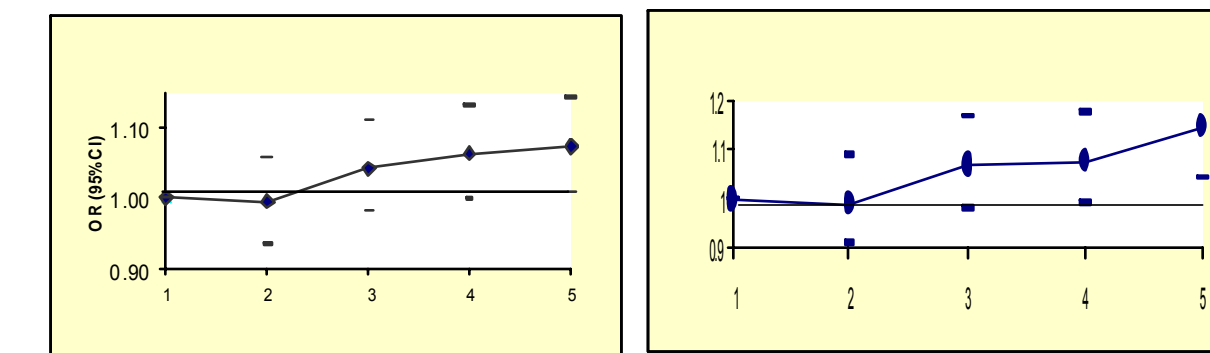
Number of freeway trucks passing within 750 feet of a home per day	Odds Ratio (95% CI)
13,290 trucks	1.23 (1.06-1.43)
8,684 heavy-duty diesel vehicles	1.18 (1.02-1.37)
Model adjusted for all maternal risk factors as covariates, background air pollution concentrations and census block-group level socio-economic status.	

Infants born between 1997-2000 in Los Angeles County

DWTD and Preterm Birth in LA: 1994-1996

(Case N=17,706; Control N=26,005)

Conclusions: In Los Angeles, risks of **term LBW and preterm birth increase with DWTD** and there are greater risks in **winter births** and areas with **higher background air pollution**.



Animal Exposures at the Freeway

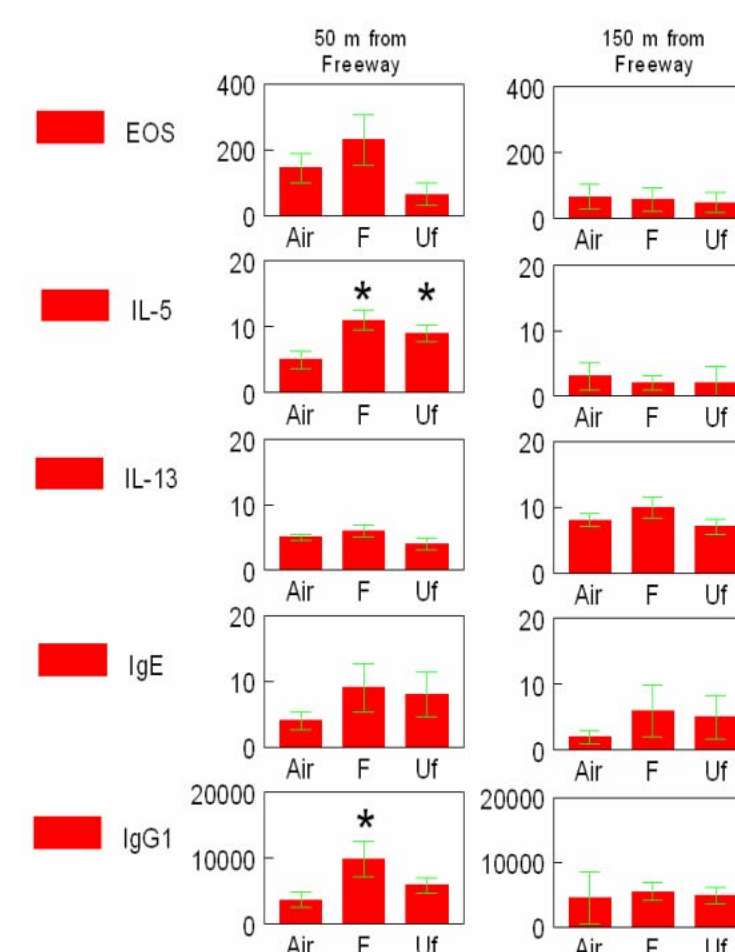
Hypotheses

- **Mobile emissions** will **exacerbate** airway inflammation and allergic airway disease, as well as affecting extrapulmonary organs (brain/heart).
- The **magnitude** of allergic airway disease responses will be **greater** at sites with **higher concentrations** of ultrafine particles.
- **Organic and inorganic** PM constituents that can generate reactive oxygen species (ROS) will be associated with responses.

Methods:

- Ovalbumin-sensitized **mice** and senescent (geriatric) **rats** are exposed to freeway-derived, concentrated **ultrafine** (UF; dp<2.5 μm) and **fine** (F; dp<0.15μm) particles at sites that are **50m** and **150m** downwind from a heavily trafficked freeway.
- **Control** animals are exposed to **purified air**.
- Particles are concentrated using a Versatile Aerosol Concentration and Exposure System (**VACES**).

Airway allergy responses



Conclusion: Exposure to CAPs **50 m** downwind of a heavily trafficked freeway caused a **pattern of responses** consistent with TH2 activation in OVA-sensitized mice.

Biomarkers of TH2 activation were elevated in CAPs-exposed OVA-sensitized mice at 50m downwind of a heavily trafficked freeway.

Significant increases compared to controls were seen for IL-5 and IgG1.

Biomarker levels in mice exposed to CAPs 150 m downwind of the freeway were not different from controls.

Brain inflammation markers

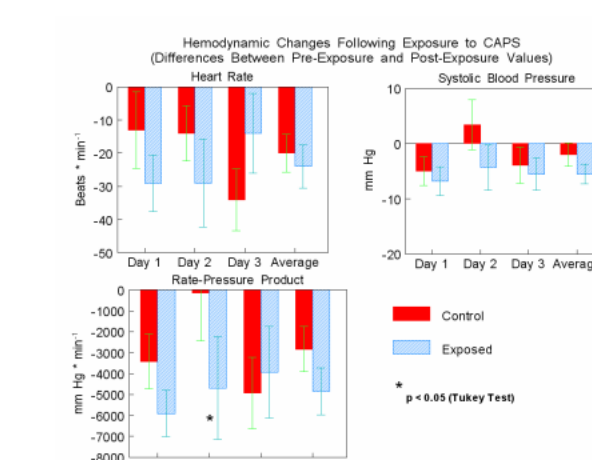
Location	Near Freeway			Receptor Site (Riverside)		
	Control	UF	F	Control	UF	F
TNF (ng/mL)	2.00.1	2.20.1	2.50.2*	NA	NA	NA
IL-1 (ng/mL)	1.60.2	2.70.3*	2.80.4*	NA	NA	NA
NFκB (% of Control)	1005	1292**	1263**	100±3	103±11	103±14
AP-1 (% of Control)	NA	NA	NA	100±12	112±16	109±30

Note: Significantly different from air control values *p<0.05; **p<0.01 NA= Not Analyzed

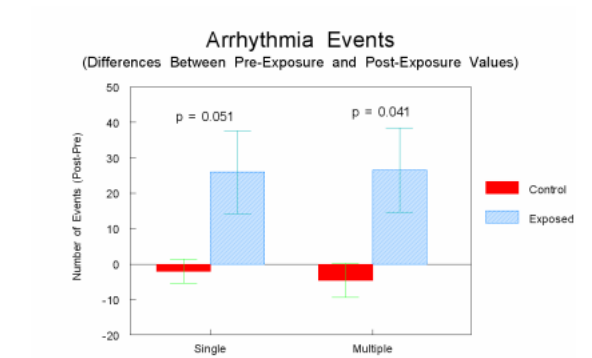
Brains from CAPs-exposed mice were assayed for inflammatory mediators. TNFα and IL-1α show infection or injury and NFκB and AP-1 can enter the nucleus of a targeted infected or injured cell and activate genes that kill the cell. Finding elevated levels of these mediators in brain tissue 2 weeks after the end of exposures suggests a persistent inflammatory effect of inhaled PM on the brain.

Conclusion: Fine and ultrafine **particles** near a heavily trafficked road produced **significant increases** in **inflammatory biomarkers**.

Cardiovascular effects of CAPs exposure in senescent rats



HR and BP showed a normal diurnal **decrease** from morning to evening in rats exposed to purified air. The pattern was exaggerated in rats exposed to CAPs. The decrease in the average Rate-Pressure Product is indicative of **decreased cardiac output**.



Conclusion: CAPs exposure can **adversely** affect **cardiac function** and increase arrhythmias in aged rats.

Impact and Outcomes

PM associated with vehicles is high in ultrafine PM that contains significant organic and elemental carbon composition (see Sioutas poster). As the distance from freeways increases, the number of particles decreases, their size grows and they change in character, raising important questions about the toxicity of the newly formed particles.

Studies conducted in Los Angeles have demonstrated **significant toxicity** and potential health impacts **from vehicle pollution**. It appears that mobile source PM is an important factor in the health effects associated with PM exposure in the urban environment. A consistent pattern of activity appears associated with mobile source pollution including new health endpoints previously unidentified.

Human studies have shown:

- Developmental toxicity;
- Impacts on child lung development from chronic exposure;
- Increased asthma prevalence in relation to distance from freeways;
- Changes in heart rate variability; and
- Lung function disturbances.

Animal models have shown:

- Neurological inflammation;
- Heart rate and blood pressure changes, arrhythmias; and
- Airway allergic responses.

These findings demonstrate that freeway-related emission problems may be **greater than previously understood** and they **raise new issues** about health effects that may impact **commuters** and persons living in close proximity to freeways.

Future Research

- Characterization of the toxicity of PM derived from vapor-phase condensation which occurs subsequent to release; Further comparisons of the relative toxicity of diesel versus gasoline emissions;
- Additional research on toxicological potency of PM as a function of distance from freeways;
- In-vehicle studies to assess impact on commuters;
- Further investigations of the toxicity of various PM size fractions with differing chemical composition; and
- Assessment of the contribution of outdoor sources to indoor exposure and toxicity.

This poster does not necessarily reflect EPA policy. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

Source to Health Outcome

*A major contributor to this work was the California Air Resources Board.